Modern Concepts of Cardiovascular Disease

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THE ROLE OF PERIPHERAL CIRCULATORY FAILURE IN CLINICAL MEDICINE

Introduction

There is an increasing recognition of the importance of peripheral circulatory failure or shock in the field of general medicine. This complication appears in a variety of disease processes, widely different in primary etiology, and when present often becomes of more serious significance than the initiating pathological state. Treatment of shock is so specific and so frequently successful that it is important to recognize it at once and differentiate it precisely from situations with which it might be confused. Consideration of surgical and traumatic shock will be omitted from this discussion, which will be confined in main to the incidence and mechanism of what may be called medical shock.

Definition and Description

Shock occurs when there is an acute disparity between the blood volume and the functioning capacity of the vascular bed. The components of the clinical picture resulting therefrom include great prostration, cold extremities, small rapid pulse, low blood pressure, weak heart sounds, sweating, anuria, pallor, and sunken eyes. This alarming clinical state may be explained by the pathological physiology of the peripheral circulation. There is stasis of blood in the capillaries, with a generalized anoxemia and tissue asphyxia. Numerous vicious circles are set in motion. Cardiac output decreases and hence further slows the circulation. The capillaries dilate, due to oxygen want, with consequent increase in the vascular bed and decrease in blood volume by leakage of plasma into the tissues. The kidneys cease to function, and electrolyte physiology is disturbed. Nitrogenous waste products accumulate. Bacterial toxins cannot be properly eliminated. The whole body economy is at a standstill during what is usually a very critical period in the disease of which shock is a complication.

Mechanism of the Production of Shock

An acute disparity between the blood volume and the vascular bed, necessary to produce shock, may be primarily due to a decrease in blood volume. This effect may be accomplished by a direct loss of whole blood, as in hemorrhage; by a loss of salt and water by a variety of routes; and, finally, by a loss of plasma from capillaries that have dilated and hence become more permeable to plasma. On the other hand, the discrepancy may depend upon an enlargement of the vascular bed by toxins that dilate capillaries either by direct physiological effect or by poisoning them. In practical experience these two mechanisms usually operate together in the production of peripheral vascular failure as a complication of many disease states.

Dilatation of the Vascular Bed

The administration of histamine in large doses will cause such marked general capillary dilatation that shock ensues. A similar situation is seen following intravenous injection of the venom of certain snakes. There is rather convincing evidence that some bacterial toxins act upon the capillaries in a similar fashion. The pneumococcus belongs to this group, and experimental results with this organism may offer partial explanation of the not infrequent occurrence of peripheral vascular failure in pneumonia. Many instances of so-called cardiac failure in very severe infectious disease are in reality due to the appearance of shock brought about presumably by the influence of bacterial toxins upon the capillary bed.

Decrease in Blood Volume

Simple hemorrhage needs no special discussion, but the mechanisms producing shock by dehydration and salt depletion deserve detailed mention. The tremendous loss of water and salt in *cholera* produces physiological results that are probably more serious to the patient than the toxins of the cholera organism itself. This fact was recognized over a hundred years ago. The mortality in cholera has been greatly reduced by treating this complication. Intense *diarrheas* from any cause have the same serious potentialities as cholera.

Severe and persistent vomiting such as occurs in pyloric or intestinal obstruction are analagous sources of salt and water loss and may be expected to produce eventually the same state of shock as does loss of these substances by way of the colon. In the case of intes-

tinal obstruction, much fluid may remain statically immobilized in the lumen of the gut, and hence as unavailable for the circulating blood stream as if it had been lost to the exterior. *Fistulous openings* to the gall bladder, stomach, pancreas, and intestine may lead to the drainage of bodily secretions high in water and salt content. Shock is the inevitable result of such losses when they are quantitatively adequate to produce a drop in the blood volume.

Severe diabetic acidosis leads to shock by two apparently unrelated physiological processes. The glycosuria alone tends to remove significant amounts of water and base, but when acidosis is a further complication, even more base is required for the renal excretion of ketone acids. Clinical experience has shown that dehydration and salt depletion are more often the forces causing a fatal issue in this disease than the state of acidosis itself; in fact, it is possible for improperly treated patients to die of shock with a normal blood bicarbonate level. The evidences of circulatory insufficiency that are found in advanced diabetic acidosis are often confused with cardiac or renal failure. Therapy, thus misdirected, is not only of no value but may indeed be harmful.

It has been shown in recent years that the serious consequences of extensive *burns* may be in part at least attributed to a decrease in blood volume. The large volumes of serous exudate contain much base and water and introduce thereby the series of events that so characteristically lead to shock. In addition to that which weeps from the burned areas, further exudate is removed from the blood stream to form the edema of the underlying tissues. This latter factor has been shown by experiment to be adequate in itself to produce shock in both burned and frozen animals.

Addison, in his original description of adrenal insufficiency, commented on its similarity to the terminal stages of cholera. Recent work has shown that the disturbances of physiology occurring in *Addison's disease* or following adrenalectomy in animals include striking effects on the sodium metabolism. This basic substance is excreted by way of the kidney in large quantities when cortical insufficiency appears. The sodium carries with it sufficient amounts of water to produce terminally a state of shock due to salt and water depletion. The beneficial effect of sodium replacement therapy gives excellent confirmation to these physiological observations.

Under long exposure to *excessive heat* the body excretes large quantities of water and salt in the form of sweat. There is evidence that the ensuing heat prostration may be considered in some degree to belong with the group in which peripheral circulatory failure, due to decreased blood volume, plays an important role.

Although emphasis has been placed upon two different types of approach to the state of shock, *viz.*, vasodilatation and decreased blood volume, only rarely does one process exist wholly independent of the other. For example, in diabetes there is some evidence which indicates that the ketone bodies may be toxic to the capillaries, thus introducing capillary dilatation as another factor contributing to the production of shock.

Sympathetic Nervous System

A mechanism entirely different from the processes described above probably operates to produce the state of shock which is seen in *cardiac infarction*. This complication occurs before any toxic vasodilating substance could be liberated, and no forces leading to a decreased blood volume exist. One is compelled to consider some influence on the sympathetic nervous system as the most probable causal factor. Certain differences of opinion exist as to the therapeutic indications of shock in cardiac infarction. On the one hand it is thought to exert a protective influence on the damaged heart, whereas another point of view urges that, whenever it occurs, stasis in the peripheral vascular bed must be harmful and should be treated as actively in cardiac infarction as it would be in other clinical conditions. It is too soon to attempt a final answer to these opposing attitudes.

Differential Diagnosis

Peripheral vascular failure is most frequently confused with cardiac failure. This is a serious mistake, because it not only precludes the use of the proper treatment, which is highly successful in most instances, but it also causes the clinician to have an incorrect orientation to all aspects of his problem. Of course, the most important aid to recognition of this complication is thorough familiarity with the underlying physiological disturbances that produce it. However, in those situations where a background of cardiac disease introduces a genuine factor of confusion, no procedure is as fruitful as a determination of the venous pressure. A degree of cardiac insufficiency sufficiently advanced to produce circulatory changes simulating shock would almost certainly have an elevated venous pressure. A low or normal venous pressure is usually found in simple shock. As a single exception one must cite the occasional occurrence of a very high venous pressure in shock due apparently to constriction of the veins.

Treatment

The treatment of shock is more or less independent of its cause. Whether it be due to trauma, toxemia, hemorrhage, or dehydration, the physiologic problem is the same; namely, a disparity between the circulating blood volume and the vascular bed. On one hand there is primarily a decreased blood volume from hemorrhage or fluid loss; on the other an increased vascular bed resulting from capillary dilatation. The need for immediate measures to increase the circulating blood volume is common to all types.

It is reasonable to say that the longer the state of shock is permitted to exist, the more difficult it becomes to alleviate it and the higher is the mortality.

Consequently, delay in initiating therapy is dangerous. As an emergency method, the intravenous injection of 50 cc. of 50 per cent glucose, which may be conveniently kept on hand, is of some temporary value, as it will draw fluid into the blood stream from the tissue spaces. Its action is usually fleeting, and, if necessary, it should be followed promptly by the intravenous injection of 1,000 to 2,000 cc. of normal saline solution. When there is no pre-existing cardiac damage, a return of the arterial blood pressure to higher levels may be accepted as a criterion of the amount of salt solution necessary for therapeutic success. There is, among clinicians, a very general belief that the intravenous administration of fluids in large quantities tends to constitute a dangerous strain on the myocardium. This fear is greatly exaggerated. However, if there is any question as to the cardiac status, a venous pressure monometer should be inserted into the infusion apparatus, and a rise in venous pressure during the injection of fluid should be used as an index of overload of the circulation. Such overload is very rare, even when several liters of fluid are necessary for satisfactory replacement. The value of salt solution in the treatment of shock developing in the course of infectious diseases has not been thoroughly tested, and final conclusions as to its usefulness are still uncertain. In shock due to other causes, dramatic results follow this therapeutic measure.

Although normal salt solution is usually an adequate therapeutic agent, it should be remembered that in many of the conditions enumerated above the salt depletion is relatively more rapid than the fluid loss. Under such circumstances it is wise to use higher concentrations of salt, such as 1.0 per cent or even 5 per cent, until the restoration of base is complete. Salt solutions of the former concentration (1 per cent) may be used interchangeably with normal solutions, but it is unwise to give more than 300 cc. of 5 per cent saline at one time.

The ideal treatment for shock is a large blood transfusion, and every patient likely to develop shock should have his blood grouped early in the course of his disease. When salt solution has failed, transfusion may turn the tide. In an attempt to find a substitute for blood, numerous colloidal substances have been tried. The most prominent of these is acacia. During the World War this solution was tried and given up because of the severe reactions which frequently followed its administration. In the past two or three years methods of preparation of acacia solutions have been improved, and satisfactory reports of its action are now appearing.

Replacement therapy in severe shock should be given *intravenously* rather than subcutaneously or intramuscularly. The rapidity of response is much greater when the intravenous route of administration is employed, because the poor peripheral circulation greatly retards subcutaneous absorption.

The use of vasoconstricting drugs, such as adrenalin, is not helpful and may indeed be harmful. From a physiologic standpoint they are contraindicated, because the blood vessels which they affect are already constricted to the disadvantage of the capillary circulation, as has been shown by studies of both the skin and visceral arterioles. Furthermore, experimental work has shown that continuous injection of adrenalin in quantities that are equivalent to the amounts produced by sympathetic stimulation can cause a 14 per cent decrease in blood volume during a two-hour period. Hence, adrenalin exaggerates the state of shock, even though it temporarily increases arterial pressure.

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